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ASEPTIC MENINGITIS CAUSED BY *COXIELLA BURNETII*

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Abstract: Acute Q fever can have multiple presentations but neurologic involvement is rare. We describe the case of a 16-year-old female with severe headache and aseptic meningitis with acute Coxiella burnetii infection.

Key Words: acute Q fever, *Coxiella burnetii*, meningitis

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Q fever is a zoonosis caused by the obligate intracellular bacterium Coxiella burnetii. Clinical manifestations of acute infection vary greatly; acute meningitis is a possible but rare form of presentation. We report the case of a patient admitted with aseptic meningitis caused by C. *burnetii* acute infection.

CASE REPORT

A 16-year-old female presented to the emergency department complaining of severe headaches (mainly retro-orbital) for the previous 6 days, with photophobia and vomiting for 2 days. She was previously healthy, and was vaccinated according to the Portuguese National Health Department recommendations. She denied regular contact with animals, although she used to visit her grandparents in a rural area, where they had 2 dogs.

On physical examination she was slightly pale, and on ophthalmoscopy the papillary edges seemed attenuated on the left eye. The remaining findings were unremarkable. Blood tests were performed, which revealed microcytic and hypochromic anemia (Hg 10.2 g/dL), no leukocytosis, thrombocytopenia (105×10^9/L), elevated C-reactive protein (107 mg/L) and erythrocyte sedimentation rate (56 mm/h) and elevated liver enzymes (alanine aminotransferase 105 U/L, alkaline phosphatase 107 U/L). The chest roentgenography showed a mild perihilar infiltrate. The cerebral magnetic resonance imaging revealed no indirect signs of intracranial raised pressure or other significant changes, and a lumbar puncture was performed. Clear, transparent cerebrospinal fluid (CSF) was obtained, with 92 cells/μL (60% polymorphonuclear), low glucose value (60 mg/dL, index CSF/serum 0.4) and elevated protein (66.3 mg/dL). Blood and CSF cultures were obtained, treatment with ceftriaxone and vancomycin was started, and she was admitted with the clinical suspicion of meningitis. She improved after 2 days and completed 8 days of antibiotic therapy. Blood and CSF cultures were sterile. She was re-evaluated after 4 weeks and remained asymptomatic, but the *C. burnetii* serology was then known to be positive (phase II IgM 1/256 and IgG 1/12; phase I IgM negative and IgG 1/64). She was again repeatedly questioned for animal exposure, and she eventually recalled attending a traditional open-air market with livestock, when she last visited her grandparents around 2 weeks before the hospital admission. Diagnosis of Q fever was assumed, and she was treated with doxycycline.

DISCUSSION

Clinical manifestations of acute Q fever vary greatly.[1–5] The most common presentations are self-limited flu-like syndrome, prolonged fever, pneumonia or granulomatous hepatitis,[1,3,5] but several distinct manifestations have been described, such as meningoencephalitis, myocarditis, erythema nodosum, pancreatitis, thyroiditis, haemolytic anaemia, epydidimitis or mediastinal lymphadenopathy.[1]

Bernit et al[2] reviewed a series of patients with acute Q fever and evidence of neurologic involvement, and concluded that the overall prevalence was 2.2%. In their series, severe headache with a meningeal syndrome was the most common clinical manifestation; encephalitis or meningoencephalitis was more common than meningitis alone; and meningitis was usually lymphocytic. In our case, pleocytosis was mainly because of polymorphonuclear cells, but this is a well-known phenomenon in the initial stages of several lymphocytic meningitis, such as viral, spirochetal, mycobacterial or fungal meningitis. In contrast to Bernit et al's[2] study, Reilly et al[6] reported a rate of 22% regarding neurologic complications in acute Q fever, which suggests that it may not be so rare.

Doxycycline is the first-line treatment of acute infection, although it has been suggested that fluoroquinolones could be a better option because of better penetration into the CSF.[2] Neurologic recovery usually occurs regardless of treatment.[2] Some studies suggest that ceftriaxone might have bacteriostatic activity against some strains of *C. burnetii*, but this is not the preferred treatment.[1] In this case report, it is impossible to know whether the patient improved as a part of the natural history of the disease or if ceftriaxone had some impact on its clinical course. We decided to treat the patient with doxycycline to prevent evolution for chronic disease.

In conclusion, acute Q fever can have a multitude of clinical manifestations, and the epidemiologic clue for exposition to *C. burnetii* is not always obvious. Although it is generally regarded as a rare occurrence, it is important to recognize *C. burnetii* as a cause of acute meningitis because the first-line treatment for this agent is not usually included in the empiric antibiotic coverage used for meningitis, and the evolution for chronic Q fever could have devastating consequences.

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